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Department of Metabolic Diseases, GlaxoSmithKline Inc., 5 Moore Drive, Research Triangle Park, North Carolina 27709, USA.

Type 2 diabetes is associated with insulin resistance in peripheral tissues, such as muscle and fat. Novel therapies that improve insulin action include ligands that bind and activate the nuclear receptors peroxisome proliferator activating receptor gamma (PPAR gamma) and retinoid X receptor (RXR). PPAR gamma/RXR form heterodimers that regulate transcription of genes involved in insulin action, adipocyte differentiation, lipid metabolism and inflammation. PPAR gamma activators include prostanoids, fatty acids, thiazolidinediones and N-(2-benzoylphenyl)tyrosine analogues. RXR ligands include naturally occurring retinoic acid and synthetic retinoids. Selective ligands for these receptors improve metabolic abnormalities associated with type 2 diabetes, such as hyperglycemia, hyperlipidemia, insulin resistance and other cardiovascular risk factors. Although adipose tissue mediates some of the effects of PPAR gamma/RXR ligands, other tissues also regulate the effects of these receptors. The activity of the PPAR gamma/RXR heterodimer is influenced by posttranslational modifications, receptor turnover, polymorphisms, splice variants, coactivators and corepressors. This article reviews recent developments in research on these receptors, with particular emphasis on metabolic effects, ligand selectivity, structure and regulation of the PPAR gamma/RXR heterodimer.

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